

Glomerular Disorders

Pathogenesis of Immune-mediated Glomerulopathy

Hypersensitivity Reactions

- Occurring within the kidney (type II cytotoxic).
- Circulating antibodies + antigens at the glomerular basement membrane (GBM) in 5% of cases of glomerulopathy.
- Example:
 - Good pasture's syndrome.
 - Rapidly progressive glomerulonephritis.

Immune-complex Disease

⇒ Type III hypersensitivity:

- Antigen (exogenous or endogenous) + antibody + complement → **initiate inflammatory process in the kidney.**
- Renal biopsy → **immune complex deposits** in the kidney.
- Examples:

Antigens	Clinical Condition
<u>Exogenous:</u> • Drugs – toxoid. • Foreign serum.	• Serum sickness. • Glomerulonephritis
<u>Bacterial infections:</u> • Streptococci. • Staphylococci. • Salmonella. • Syphilis	• Post- streptococcal GN. • Typhoid fever. • Membranous nephropathy.
<u>Parasitic infections:</u> • Malaria • Bilharziasis. • Toxoplasmosis.	• Nephrotic syndrome.
<u>Viral infections:</u> • Hepatitis B. • EB virus.	• Viral hepatitis + rapidly progressive GN.
<u>Endogenous:</u> • Nuclear antigens. • Tumor antigens. • Renal tubular antigens.	• SLE nephropathy • Hodgkin's + GN.

Initiation of Cellular Immunity by GBM antigens

Example: chronic GN.

Clinical Presentations Of Glomerular disorders

1. Nephrotic syndrome.
2. Acute GN.
3. Nephritic Nephrotic.
4. IgA nephritis.
5. Acute renal failure (ARF).
6. Chronic renal failure (CRF).
7. Recurrent or persistent hematuria.
8. Asymptomatic proteinuria.
9. Rapidly progressive GN (with crescent)
10. Hemolytic uremic syndrome (HUS).

Nephrotic Syndrome

Definition

- It is a clinical syndrome characterized by:

- 1- Heavy proteinuria > 2 gm/24hrs.
- 2- Hypoproteinemia.
- 3- Marked generalized edema.
- 4- ± Hypercholesterolemia.

Etiology

1ry	2ry
1. Minimal change GN. 2. Cong. GN. 3. Membranous GN. 4. Membrano-proliferative. GN. 5. Rapidly progressive GN. 6. Focal glomerulosclerosis.	1. SLE. 2. DM. 3. Henoch schonlein purpura. 4. Sickle nephropathy. 5. Malignancies e.g. lymphoma. 6. Gold & D-penicillamine. 7. Malaria & S. Mansoni.

→ auto Ab against
sialophr.

Minimal Change GN

Etiology

↳ only pathology in ! kid. is fusion of foot processes.

- Loss of -ve charge of sialoproteins which are present in the wall of fenestra of basement membrane due to immunologic disorders (T-cell dysfunction) so, it is improved by measles infection. ⊗ Bad.

Incidence

1. > 70% (commonest cause of nephritic syndrome in pediatrics).
2. Age: 2-7 Years (it can be < 1 year).
3. Sex: ♂ : ♀ = 2 : 1.

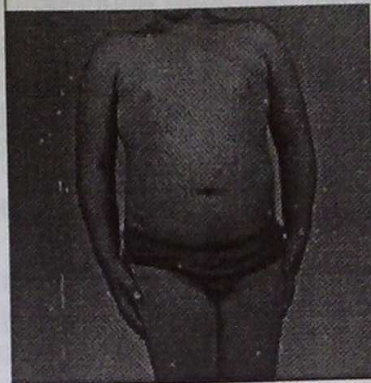
Clinical Picture

1. Massive Generalized Edema:

- Puffy eye lids.
- Pleural effusion
- Pericardial effusion → *periCard. الـسوائل*
- Ascites.
- Scrotal edema
- Lower limb edema.



Puffy eye lids

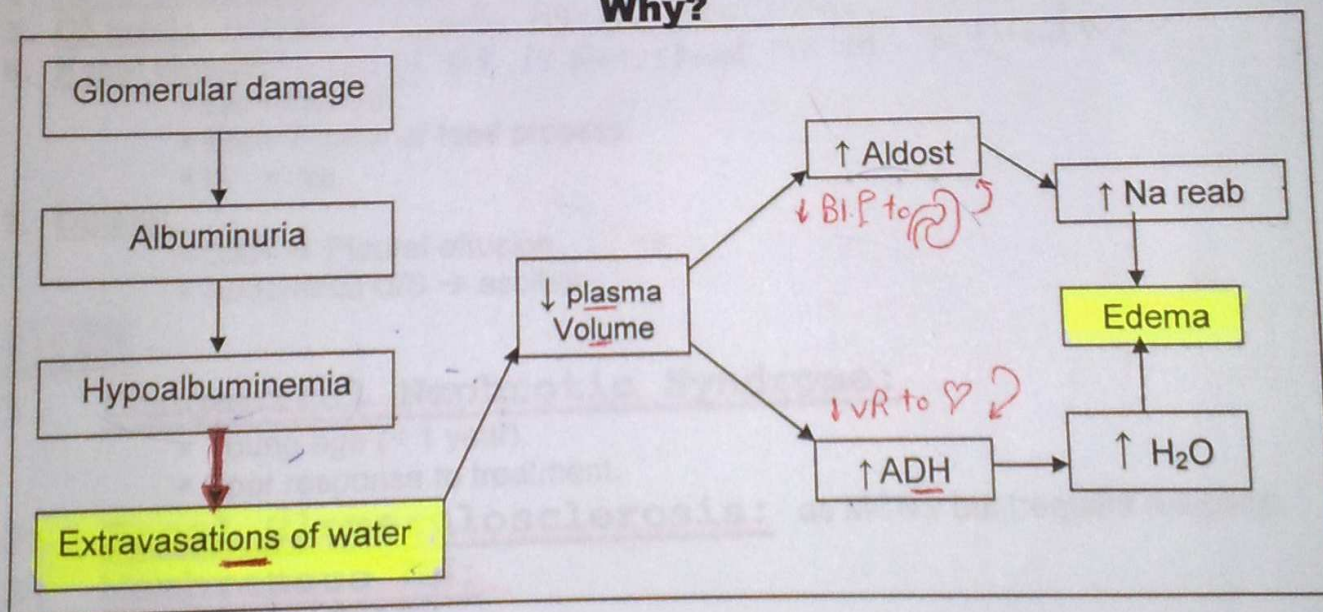


Ascites



Scrotal edema

Why?



2. No:

- Oliguria
- Hypertension
- Hematuria

3. Associated Complications:

A. ↑↑ Tendency for Infection due to:

1. Loss of IgG.
2. Loss of tuftsin. *"acts against capsulated org. " Phagocytic functions.*
3. Immunosuppressive drugs used in treatment.
4. Presence of edema or ascites. *"wetness - stasis"*

B. **↑↑ Risk of Thrombosis due to:**

- ④ **↑ B. Viscosity**
1. Hemo-concentration due to edema & use of diuretics., *Steroid d.t Poly Cythermia.*
 2. Hyper-cholesterolemia.
 3. Loss of antithrombin III, protein C&S in urine. ↓
 4. ↑↑↑ release of certain coagulation factors. ↑

Investigations1. **Urine analysis:**

- Heavy proteinuria (> 2gm /24hrs urine).
- Mainly selective proteinuria (albumin, transferrin, IgG).

2. **↓↓ Total Protein & Serum Albumin < 2.5 gm/dl.**

- Normal level of protein = 6-8 gm% & albumin = 4.5-5 gm %).

▪ Hypoproteinemia is due to:

- 2ry to excess urinary loss.

- Loss of serum protein into the GIT. *edema in mm of GIT*

- (*) **↑↑ rate of albumin catabolism.** *"congestion"*

3. **Serum Cholesterol:**

- > 300 mg/dl (normal level = 150-250 mg %).

▪ Hyperlipidemia is due to:

- **↑↑** lipoprotein synthesis by the liver to correct the decreased osmotic pressure 2ry to Hypoalbuminemia.

- **↓↓** destruction of cholesterol by the liver due to **↓↓** lipoprotein lipase enzyme.

4. **Renal functions:** normal.5. **C3 level:** normal.6. **Renal biopsy:**

- LM = Normal.
- EM = Fusion of food process.
- IF = -ve.

7. **Others:**

- CXR → Pleural effusion.
- Abdominal U/S → ascites.

DD

1. **Congenital Nephrotic Syndrome:**

- Young age (< 1 year).
- Poor response to treatment.

2. **Focal Glomerulosclerosis:** as MCNS but frequent relapses.3. **Membranous GN:**

- Age > 10 years.
- Non-selective proteinuria.
- Poor response to treatment.

4. **Membrano-proliferative GN:**

- Age > 10 years.
- **↓↓↓** C3.
- Hematuria.
- Hypertension.
- Azotemia.

5. **Secondary glomerulopathy with systemic diseases:**

- DM & SLE.

Treatment

1. Rest:

Because exercise will lead to

1. ↑↑ proteinuria ↑ $\uparrow$ R, upright position → ↑ Renal Bl. flow → ↑ GFR, ↑ urea.
2. ↑↑ catabolism of protein. "↑ BMR".

2. Diet:

1. High albumin containing food e.g. egg - white.
2. Low salt diet. → as Aldosterone is ↑ + salt → aggravate oedema.
3. Low fat.
4. ↑↑ CHO. → يحرقها بدل البروتين

3. Diuretics:

Usually given alone in the 1st two weeks without steroids because:-

1. Spontaneous remission may occur.
2. ↓↓ Ascites → ↑↑ renal perfusion. → ↓ Compression on Kidney.
3. ↓↓ Edema → so we can reach the actual body weight.
4. ↓↓ Tissue edema → enhance response to steroids.
5. During these 2 ws we can exclude any infections esp. T.B.

One of the following drugs can be used

1. Lasix (furosemide) → 1-3 mg / kg + K syrup
2. Aldacton (spironolactone) → 1-3 mg / kg.
3. Osmotic diuretics → Mannitol 20% IV drip over 30 min followed by IV Lasix 1 mg / kg.
4. Salt free albumin followed by lasix: To ⊖ hypervol., ⊖ congestive HF.

The role of salt free albumin in treatment is:

- Strong diuretics in presence of massive edema.
- It compensates ↑↑↑ level of lipoproteins. ↑↑↑ level of big molecular weight proteins, ↑↑↑ level of coagulation factors.

4. Drugs:

- Prednisone 2 mg / kg / D (max. 60 mg / m² / D). →
- Cyclophosphamide (Endoxan): 1-2 mg / kg / D for 6 ws.
- Chlorambucil in frequent relapses 0.2-0.4 mg / kg for 3 months.
- Indomethacin (Indocid) in resistant cases.
- Azathioprine (Imuran).
- Levamisole (Ketrax): 2.5 mg / kg twice / week.
- Anticoagulants in cases of membrano-proliferative type or in presence of thrombosis.
- Cyclosporine A (5 mg / kg) for 6 months in resistant cases or presence of side effects of steroids.

Treatment

A. Treatment of 1st Attack:

- The aim of Treatment is to ↓↓ protein excretion in urine.
- Usually we start by prednisone tablets 2 mg/kg/D for 10-14 days after complete absence of proteinuria, and then start gradual tapering.
- If proteinuria persists after one month, it is an indication for renal biopsy.
- According to renal biopsy:

- ⇒ Minimal change NS: give prednisone for another one month.
- ⇒ Focal glomerulosclerosis: Intermittent prednisone + alkylating agents for 3 months.
- ⇒ Membranous nephropathy: no Treatment (10% pass to chronic renal failure).
- ⇒ Membrano-proliferative GN: intermittent prednisone therapy + anticoagulant + Antimetabolite (azathioprine).

نظروا على نتائج
urine

3-6

Bad Prog.

Permeable
Re biopsy
on table

- 30 % of patients who respond to steroids will not have relapse & 70 % will relapse for 1-3 times/ year. *بنت ساء الله*

B. Treatment of Relapse:

- Start with prednisone as Treatment of 1st attack, then give steroids one single dose in the early morning every other day for 6 months. *6w → 3ms*

C. Treatment of Frequent Relapses:

- Intermittent prednisone + daily Chlorambucil or Cyclophosphamide for 6 weeks to 3 months. *Clinical WQ*

Nephrotic Syndrome in the 1st Year of Life

1. Infantile micro cystic disease (congenital nephrosis):

- Autosomal recessive disorder. *< 1 y*
- Clinically presented by generalized edema & highly selective proteinuria since birth. *→ diagnostic*
- Renal biopsy: cystic dilatation of proximal renal tubules. *فالتشخيص*
- Can be diagnosed intra-uterine by detection of $\uparrow\uparrow$ α -fetoprotein in amniotic fluid at 14-16 weeks.
- The prognosis is usually fatal ending in renal failure. *THH: No SPotransplant*

2. Membranous nephropathy of congenital syphilis:

- Onset at 6 months. *Common at 10 y age.*
- Renal biopsy: membranous nephropathy immune deposits. *BAK → non selective pturia*
- Antisyphilitic treatment is curative. *No Response To THH*

3. Minimal change Nephrotic syndrome: Can occur at the age of 6 months.

4. Focal glomerulosclerosis.

5. Idiopathic membranous glomerulopathy:

- Treated by intermittent course of steroids for 6 months.

6. Mercury poisoning.

7. Congenital toxoplasmosis: treated by daraprim 2 mg/ kg.

8. Nail-patella syndrome:

- Nephrotic syndrome + Absence of patella + Atrophy of nails.

9. Nephroblastoma: with or without abnormal genitalia.

10. Hemolytic uremic syndrome:

- Autoimmune disease $\Rightarrow$ Acute hemolytic anemia + massive nephritis + thrombocytopenia + microangiopathy. *see Blood*
- It is a serious disease usually preceded by a short period of gastroenteritis or respiratory tract infection. Then after 2 days the infant develops sudden pallor, Oliguria or anuria, toxemia & bleeding tendency in the form of petichae or purpura.
- It is an example of consumption coagulopathy & characteristic RBCs $\Rightarrow$ Burr cells.
- Treatment by plasmapheresis or urgent peritoneal dialysis.

Acute Post-streptococcal GN

Etiology

- It is an immune-complex disease.
- Due to infection by group A β hemolytic streptococci:
 - Pharyngeal strain 12: more in winter.
 - Skin strain 49: more in summer.

43

(5)

Incidence

- Age:** 5-15 years.
- Sex:** more in males.
- Course:** 2-3 weeks.

الوادر
بعد الحماكة
بالمسوخ

Clinical Picture

History

- Of streptococcal infection 1-3 weeks ago.

The Classic Criteria of Nephritic are:

- Hematuria.
- Oliguria $< 400\text{ml} / \text{m}^2$
- Hypertension
- Edema "mild".

1. Hematuria & Oliguria:

- Dark urine + diminished urinary output is the most frequent manifestation.
- In the first few days the urine is grossly bloody then becomes smoky brown.

في الاول يكون
غامق اوى وبنه
بيبقى اخف



2. Edema:

- Usually mild in the form of puffy eyes & edema of extremities.
- Due to:

- $\downarrow\downarrow$ GFR.
- Vasculitis $\rightarrow$ toxic capillaritis.
- $\uparrow\uparrow$ Aldosterone $\rightarrow$ salt & water retention.

- However edema may be generalized in 3 conditions.

- Associated nephritic syndrome.
- HF.
- Renal failure without restriction of fluid intake

dorsum of hands and
feet.

3. Hypertension: "70% of cases"

- Due to:

- $\downarrow\downarrow$ GFR $\rightarrow$ hypervolemia.
- Vasculitis $\rightarrow$ thickening of the afferent A $\rightarrow \uparrow\uparrow$ rennin & aldosterone.

Complications

In severe cases any of the following 3 complications may occur.

1. Acute renal failure:

- Usually mild & transient in most cases.

2. Heart failure

- Due to:

- Hypertension.
- Circulatory overload.
- Toxic carditis.

3. Hypertensive encephalopathy:

- Severe headache.
- Lethargy.
- Convulsions & coma.

Investigations

1) Urine Analysis:

- Volume: ↓↓.
- ⊙ RBCs casts (diagnostic). → لا رقيقة قشرية لحواء
- Proteinuria (mild).
- Specific gravity: ↑↑.

2) Renal functions:

- Blood urea nitrogen (BUN): ↑↑.
- Serum creatinine: ↑↑.
- Serum K⁺: ↑↑.

3) Evidence of Streptococcal Infections:

- Anti-streptolysin-O titre (ASOT): ↑↑. (in pharyngitis).
- Anti-hyaluronidase: ↑↑. (in impetigo). skin lesion "But ASOT may be Normal"
- C3: ↓↓
- Immune complexes in the blood: ↑↑..

4) Renal Biopsy:

- Light microscope: diffuse proliferative GN.
- Immuno-fluorescent microscope: lumpy deposits.
- Electron microscope: sub-endothelial deposits.

DD

A. From other causes of GN:

1. Alport syndrome (hereditary nephritis):

- AD. sensory neural.
- GN + nerve deafness. + keratoconus
- Very young age & rapidly fatal.

نقص كريات الدم الحمراء Nephritic disease → ١٩
...! ? ...

2. IgA nephropathy (Berger's disease)

- Recurrent hematuria.
- Normal C3. → Cause is not immune complex.
- IgA renal deposits.
- ↑↑↑ IgA in the blood.

"Synpharyngitis" may lead to vasculitis.

3. Anaphylactoid purpura. "Henoch-Schönlein"

4. Rapidly progressive GN: renal failure within 2-3 ms.

5. Hemolytic uremic syndrome:

- Pallor due to acute hemolytic anemia.
- Uremia (Oliguria & Azotemia).
- Thrombocytopenia.

B. Form other causes of hematuria:

Idiopathic hematuria or benign hematuria:

- No proteinuria.
- No Azotemia.
- No hypertension.
- Normal C3 & ASOT.

C. From other causes of edema.

D. From other causes of hypertension.

Treatment

1. Early Antibiotic Therapy:

- For streptococcal infection for 10 days.
- Dose: 400,000 U procaine penicillin. IM / 12hrs after sensitivity test.

2. Fluid Restriction:

- Volume of urine in 24 hrs + 400 ml / m² / D.

3. Diet:

- ↓↓↓ protein 0.5 gm/kg/D (may reach 2 mg).
- ↓↓↓ K → if there's RF:
- ↓↓↓ Na → if there's hypertension.
- ↑↑↑ Carbohydrate.

* ↓ Fat.

4. Treatment of Hypertension:

Antihypertensive drugs كفاية

- Hypertensive encephalopathy: diazoxide 5 mg/Kg/ D IV+ lasix 1 mg / kg or propranolol 1 mg / kg or hydralazine 0.1 mg/ kg IM.
- Mild or moderate hypertension: Oral diuretics as lasix ± Hydralazine or methyldopa 20 mg / kg / D.

5. Hyperkalemia "Normally K = 3.5 → 5 meq. / L"

- Glucose + Insulin 0.1 U/kg IV drip → will enter K⁺ intracellular.
- Ca gluconate 0.5 ml / kg I.V.
- Ion exchange resin 25 gm + 5 % glucose 100 ml rectal enema.

7 - 10 mg/kg
dialysis lasix 1 mg/kg
Arrest. جفاف الكلى

6. Peritoneal Dialysis:

Indications:

- Persistent Hyperkalemia.
- Severe acidosis.
- Pulmonary edema.

* We give glucose & insulin For Risk of hypoglycaemia.

→ K enters to ! sm. ms of Bronchi → so give B₂ against To
in nebulizer → avoid Broncho Constriction.

* Ca⁺ gluconate :- gluconate + K → Bile.

Ca⁺ → ↓ Tachy arrhythmia • cardio protective ..

Acute Renal Failure

Definition

- It is a clinical syndrome characterized by **sudden reduction of glomerular & tubular functions**.

- Oliguria or anuria ($< 180 \text{ ml} / \text{m}^2 / 24 \text{ hrs}$).
- Metabolic acidosis.
- Impaired excretion of waste products such as creatinine, urea, K^+ and phosphate.
- Hyperkalemia.

Etiology

1. Pre-renal (Hypovolemia)

- Dehydration.
- Hge. *2 anasthesia*
- Shock.
- Post surgery.
- Cardiac surgery.
- Nephrotic syndrome.

⇒ Hypertension.

2. Renal

- Anoxic tubular necrosis.
- Glomerulonephritis acute or chronic:
 - Post streptococcal.
 - SLE.
 - Henoch Schonlein purpura.
 - Nephrotoxins, anesthesia, mercury and carbon tetrachloride.
 - Rapidly progressive GN.
 - Hemolytic uremic syndrome.
- Congenital abnormalities: renal agenesis or hypogenesis.
- Acute pyelonephritis.
- Tubular obstruction: uric acid nephropathy & sulfonamide crystals. *Renal cause of pr. inside Bowman's capsule.*
- Vascular:
 - Malignant hypertension.
 - Renal vein thrombosis or DIC. *aff. both.*
- Severe infection.

⇒ Hypertension.

3. Post-renal

- Congenital obstruction.**
 - Posterior urethral valve.
 - Pelvi-ureteric junction obstruction.
- Acquired obstruction:**
 - Bladder neck obstruction.
 - Ureteral obstruction. *B. Inf.*
 - Urethral stricture or stone.

⇒ Hypertension.

Causes in the Newborn

- Peri-natal anoxia.
- RDS
- DIC
- Severe hemorrhage.
- Severe dehydration.
- Septicemia.
- Congenital obstruction
- Acute pyelonephritis
- Renal vascular thrombosis.
- Hemolytic uremic syndrome.

Clinical Picture

Tetrads

- Oliguria or Anuria.
- Metabolic acidosis
- Azotemia
- Hyperkalemia

1. Anuria or Oliguria.
2. Acidosis.
3. Anorexia & pallor.
4. Dyspnea.
5. Uremic breathing.
6. Impaired level of consciousness up to coma.
7. Irritability up to convulsions.
8. Bleeding tendency due to platelet dysfunction.

Investigations

1. Urine:

- Volume: < 400 ml / m² / 24 hrs.
- Specific gravity: low fixed 1010.
- Casts: RBCs & epithelial casts.
- Na:
 - < 30 mEq/L in pre-renal failure.
 - > 30 mEq/L in renal failure.
 - > 70 mEq/L in tubular dysfunction.

2. Blood Chemistry:

- Na⁺: dilutional hyponatremia.
- K⁺: Hyperkalemia "main cause of death".
- S. Urea & creatinine: elevated.
- ABG: Metabolic acidosis.
- Uric acid: ↑↑.

But Na itself is higher than normal

3. ECG:

- Hyperkalemia.
- High peaked T-wave.

4. For the Underlying Cause.

Treatment

Measurement of blood pressure.
Initial bladder catheterization to exclude obstruction.

1. Pre-renal

- Urgent correction of Hypovolemia or shock if present by Ringer's lactate 20 ml / kg within 15 minutes ⇒ ↑↑ volume of urine, if no ↑↑ in volume of urine give mannitol 0.2 gm / kg IV within 2 minutes + lasix 1 mg / kg IV: if diuresis occurs ⇒ reversible case, if not ⇒ irreversible.

*mannitol or lasix
④ IV fluid*

2. Post-renal

- Nephrostomy
- Then → surgery.

permanent RP. 11

3. Renal

1. Restriction of fluids: urine volume in the last 24 hrs + 400ml / m² / 24hrs.
2. Treatment of Hyperkalemia: as post-streptococcal GN.
3. Treatment of hypertension if present as post streptococcal GN
4. Dialysis:
 - Severe metabolic acidosis.
 - Severe Hyperkalemia
 - Severe circulatory congestion.

Other Indications of Dialysis

- BUN > 125 mg/ dl.
- Severe hypo or hypernatremia.
- Chronic renal failure with severe symptoms.
- Poisoning by certain drugs as barbiturates, salicylates & amphetamine.
- Hemolytic uremic syndrome.

Chronic Renal Failure

Definition

- It is a clinical state in which the kidney can't maintain body homeostasis.

Etiology

⇒ In children < 5 years:

1. Renal hypoplasia. → Atrophic موجوده بس
2. Renal dysplasia. → متن موجوده اساساً
3. Obstructive uropathy. → Back pressure
4. Urinary tract malformation. ↗

⇒ In children > 5 years:

A. Hereditary:

1. Alport syndrome.
2. Polycystic kidney.

B. Acquired:

1. Glomerulonephritis.
2. HUS. → Hemolytic uremic \$

Clinical Picture

Uremic Manifestations

1. Headache.
2. Fatigue.
3. Anorexia & vomiting.
4. Lethargy & lack of concentration.

Urine Volume

1. Early in the disease:

- There is polyurea & polydipsia due to urinary concentration defect.

2. Late in the disease:

- Oliguria develops due to loss of a significant number of nephrons. This is usually followed by anuria due to complete loss of all nephrons.

Air Hunger (Rapid Deep Respiration)

- It is caused by acidosis due to:

1. Loss of HCO_3 in urine.
2. Failure of excretion of acidic materials.

Complications of Uremia**1. Bone deformities & short stature (Renal osteodystrophy):**

See renal rickets (notebook of nutritional disorders).

2. Growth failure: due to:

- a. Renal osteodystrophy.
- b. Restriction of protein in diet.
- c. Chronic anemia.
- d. Uremia $\Rightarrow$ end organ resistant to GH.

3. Pallor: due to:

- a. $\downarrow\downarrow\downarrow$ Erythropoietin.
- b. Bleeding tendency.
- c. Inadequate iron & folate intake.
- d. Urea & other toxins $\Rightarrow$ hemolysis of RBCs.
- e. Aluminum intoxication $\Rightarrow$ BM suppression.

4. Cardiovascular complications:**a. Hypertension:** due to:

- Over production of rennin.
- Na^+ & water overload.
- Hyperlipidemia due to diminished activity of lipoprotein lipase activity

b. Peri-carditis: due to deposition of uremic toxins. *may lead to effusion d.t hypervol.***c. Heart failure:** due to:

- Toxic myocarditis due to uremia.
- Hypertension.
- Anemia.
- Volume over load.

• $\heartsuit$ Tamponade d.t Pericardial effusion.

5. Bleeding tendency: due to:

- a. Thrombocytopenia.
- b. Thrombasthenia.
- c. Coagulopathies.

6. Recurrent infections: due to suppression of immune system by uremic toxins.**7. Neuropsychiatric manifestations:** due to:

- a. Uremic toxins.
- b. Hypertension.
- c. Aluminum intoxication.
- d. Trace elements & vitamins deficiencies.

d.t dyspepsia

Manifestations Related to Underlying Etiology.**Investigations****1- Urine Analysis:**

- Polyuria.
- Low fixed specific gravity 1010

2- Urea & creatinine: elevated.**3- CBC:** normocytic normochromic anemia.

Plasma

4-Electrolytes:

- Hyperkalemia. *end stage*
- Hyponatremia.
- Hypocalcaemia.
- Hyperphosphatemia.
- ↑↑ Alkaline phosphatase.

• uric acid ↑

5-Blood gases: metabolic acidosis.**6-Echo-cardiography:** for evaluation of the cardiac functions.**7-Assessment of bone status:**

- ↑↑↑ PTH.
- Sonar & scan for the parathyroid gland.
- Bone densitometry.

8-Assessment of nutritional status:

- Serum albumin.
- Serum transferrin.
- Serum zinc.
- Serum iron.
- Serum folic acid.
- Lipid profiles.

9-Investigation to detect the etiology.**Treatment****1. Treatment of the cause****2. Diet:**

- Caloric supply:** should be made to equal or exceed (in patients with growth failure) the recommended daily caloric allowance for age. This achieved by adding to diet unrestricted amounts of CHO & fat (usually medium chain triglycerides) as tolerated by the patient.
- Proteins:** if there is uremic symptoms 1.5gm/kg/day. → *give C line*
- Routine supply of water soluble vitamins especially in patients under dialysis.
- No need for supplementation with fat soluble vitamins.
- Trace element supply (zinc, iron): should be added after deficiencies are confirmed.
- Calcium.**
- Phosphate** in diet & and use calcium carbonate as phosphate binder that enhance fecal excretion of calcium phosphate.
- Antacids** containing aluminum should be avoided because of aluminum toxicity.
- Salts** if there is hypertension, volume overload or congestive HF. But ↑↑ salt may be needed in salt looser types.
- K intake & oral K-chelating resin** that binds to & remove potassium from the intestine. *only when urine volume ↓↓ "end stage"*
- Water restriction** is usually needed in patients with end stage renal disease.

3. Treatment of hypertension & uremic cardiomyopathy:

See AGN.

4. Treatment of acidosis: oral NaHCO_3 is needed if serum level falls below 20 mEq/L.**5. Treatment of rickets:** See renal osteodystrophy in notebook of nutritional disorders.

6. Treatment of anemia:

Hb level should be stabilized at 10 gm% by:

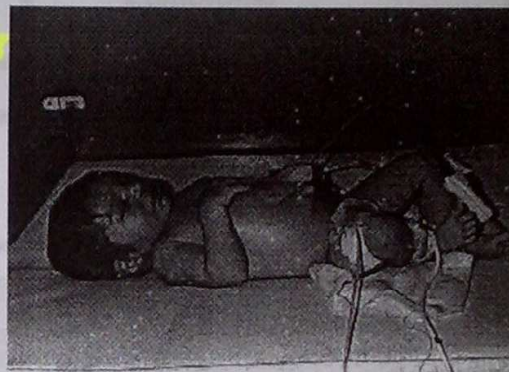
- Treatment of iron deficiency.
- Treatment of folic acid deficiency.
- Treatment of zinc deficiency.
- Treatment of aluminum intoxication.
- Treatment of infections.
- Treatment of bleeding focus.
- Erythropoietin $\rightarrow 75\text{U/kg}$.

7. Dialysis:

- Either peritoneal or hemodialysis.
- It is indicated when $\text{GFR} < 10\text{ml / minute}$. However, some patients need dialysis at earlier stages, as they can not tolerate the uremic state.

- Peritoneal dialysis:-

- a. Continuous ambulatory peritoneal dialysis.
- b. Continuous cyclic peritoneal dialysis using cyclor machine.



8. Renal transplantation.

9. Treatment of concomitant complications.

10. Social & psychological support.

11. Drug dosage:

- All drugs excreted by the kidney their doses should be adjusted according to renal function to minimize the risk of toxicity.

Urinary Hues

والتي تسمى
اللون
oval 4

Dark yellow أعمق شوية من الطبيعي

1. Concentrated urine.
2. Bile pigments.

Red urine

1. Hematuria. *e.g. GSD .. وأل فصول*
2. Hburia.
3. Urates.
4. Myoglobin. *مربيع خابطاه مثلاً Crush*
5. Porphyrins.
6. Foods as beets, blackberries, colorings.
7. Drugs: Rifampicin, chloroquine.

Dark Brown or Black

RBCs cast.

1. Homogentisic acid.
2. Melanin.
3. Methemoglobinemia.

Localization of Hematuria

	From kidney	From lower urinary tract
1. Color of urine	Brown or cola-colored.	Red
2. Relation to urine stream		May be terminal (bladder) or initial (urethra)
3. RBCs morphology	- <u>Dysmorphic</u> . - RBCs <u>casts</u> .	- <u>Eumorphic</u> - May be <u>clots</u>
4. Proteinuria	>or = 2+	< 2+
5. Associated manifestations	- Edema. - Hypertension. - Impaired renal function	- <u>Dysuria</u> . - Suprapubic pain. - Disturbed urine stream.

Hematuria

Definition

- The presence of blood in urine which may be gross or microscopic (**>5 RBCs/HPF**). *High power field.*

Etiology

A. Glomerular:

- Acute glomerulonephritis**: as acute post-streptococcal GN.
- Recurrent gross or persistent microscopic hematuria**:

- IgA nephropathy (Berger's disease):

- More common in males.
- May be high serum IgA.
- Normal C3.
- There is mesangial IgA deposits.

- Alport \$:

- AD or XLD.
- Age: 6 years.
- Hematuria.
- Sensory-neural deafness..
- Cataract & keratoconus.
- Progress rapidly to CRF.

- Membranous & membrano-proliferative GN.

- SLE.

- Rapidly progress GN.

- Nephritis of chronic infection: as in SBE, \$, malaria, hepatitis B. In all of these diseases there is high level of circulating immune complexes that deposit in the glomeruli.

- Hemolytic-Uremic syndrome: Caused by Vero toxin producing E-coli.

- Hemolytic anemia.
- Thrombocytopenia.
- ARF.

- Anaphylactoid purpura (Henoch Schonlein purpura).

*No minimal change
No ang.*

B. Non-glomerular:

1. Congenital anomalies:
 - Polycystic kidneys.
 - Vascular anomalies.
2. Infections: bacterial, viral, bilharzial.
3. Stones.
4. Tumor. *Wilms or infiltration*
5. Trauma.
6. Drugs: Cyclophosphamide & anti-coagulants.
7. Exercise.

CIN DUT

Laboratory Work-up

Phase I (for All Patients)

1. Urine analysis & culture.
2. Collection of 24 hrs urine for:
 - a. Protein.
 - b. Calcium ($n < 4\text{mg/kg/D}$).
 - c. Creatinine clearance.
3. CBC.
4. Streptococcal Abs titre.
5. Pelvi-abdominal U/S.
6. Plain X-ray on the UT & IVU.

Phase II (In Selected Patients)

1. C3 level.
2. ANA.
3. Sick cell screening.
4. Coagulation profile.
5. Ascending urethrography.
6. Audiometry.

Phase III (Invasive)

1. Renal scan.
2. Cystoscopy.
3. Renal biopsy.

Treatment

1. Supportive Treatment if hematuria is severe.
2. Treatment of the cause if possible.

*و فریبست
فیبرین*